

## Nicotine Effects Surface Bound Enolase on *Streptococcus mutans* and Its Binding to Human Plasminogen

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*Streptococcus mutans* is the major bacterial agent responsible for dental caries. Previous research has shown that smokers have increased caries and that nicotine increases biofilm formation of *S. mutans*. *S. mutans* is also associated with atherosclerosis, another disease commonly found in smokers. However, little research has been done to investigate the direct effect of nicotine on the ability of *S. mutans* to bind to endothelial cells and lead to atherosclerosis. The two objectives of this study were to determine how nicotine affects the level of enolase, a glycolytic enzyme, on the surface of *S. mutans*, and next to determine its effect on binding of treated bacteria to human plasminogen, a protein present in the bloodstream. *S. mutans* strain UA159 was grown overnight in tryptic soy broth treated with 0, 0.5, 1, and 2 mg/mL nicotine at 37°C in 5% CO<sub>2</sub>. These cells were used to coat a microtiter plate, and various levels of surface bound enolase and binding to plasminogen were determined using enzyme-linked immunosorbent assays (ELISA). A preliminary trial showed increase in both surface bound enolase and binding to plasminogen with increasing nicotine concentration. Similar results are to be expected with repetition of this procedure, indicating that nicotine up-regulates the bacterial expression of enolase and its binding to plasminogen, probably through plasminogen binding receptors, contributing to the virulence of *S. mutans*. Knowledge of the attachment mechanisms of *S. mutans* in the presence of tobacco may aid in prevention of tobacco-related atherosclerosis.

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